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Decomposing the Causes of the Socioeconomic Status (SES) Health Gradient With Biometrical Modeling

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S. Mason Garrison and Joseph Lee Rodgers
Vanderbilt University

The consistent relationship between socioeconomic status (SES) and health has been widely covered in the media and scientific journals, which typically argue that physical-health inequalities are caused by material disadvantage directly or indirectly (e.g., chronic environmental-stress, health care resources, etc.). Such explanations do not explain the finely stratified health differences across the entire range of SES. Recent theories have helped address such limitations, but implicate multiple different explanatory pathways. For example, differential epidemiology articles have argued that individual differences are the "fundamental cause" of the gradient (Gottfredson, 2004). Alternatively, variants of allostatic load theory (McEwen & Stellar, 1993), such as the Risky Families model (Repetti, Taylor, & Seeman, 2002) implicate the early home-environment. These theory-driven pathways align with interpretations associated with biometrical models; yet, little research has applied biometrical modeling to understanding the sources of the gradient. Our study presents several innovations and new research findings. First, we use kinship information from a large national family dataset, the NLSY79, whose respondents are approximately representative of United States adolescents in 1979. Second, we present the first biometrical analysis of the relationships between SES and health that uses an overall SES measure. Third, we separate physical and mental health, using excellent measurement of each construct. Fourth, we use a bivariate biometrical model to study overlap between health and SES. Results suggest divergent findings for physical and mental health. Biometrical models indicate a primarily genetic etiology for the link between SES and physical health, and a primarily environmental etiology for the link between SES and mental health.

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Keywords: socioeconomic status, SES-health gradient, behavior genetics, health inequity, NLSY

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The relationship between socioeconomic status (SES) and health has been widely covered in the popular media; within the last year, *Atlantic* (e.g., Khazan, 2017), the *New York Times* (e.g., Porter, 2017), and *Time Magazine* (e.g., Park, 2017), have featured the SES-health gradient. Both popular and scientific articles draw strongly upon conventional explanations that material disadvantage directly (e.g., through access to medical care; Hummer, Rogers, & Eberstein, 1998) or indirectly (e.g., through chronic environmental stress; Baum, Garofalo, & Yali, 1999; McEwen & Stellar, 1993) causes physical health inequalities. Such explanations account for differences between those who have resources and those who do not.

Those situational explanations, however, do not account for the finely stratified health differences that exist across the entire range of SES. They do not distinguish between mental and physical health, and in some cases even assume that the pathways are the same. In some settings, mental health is treated as a mediator, rather than as an outcome (Adler et al., 1994). Further, these explanations do not explain why greater access to health care (Siddiqi & Hertzman, 2007; Steenland, Henley, & Thun, 2002) accounting for rates of morbidity and mortality (Steenland et al., 2002), and improved education (Conti, Heckman, & Urzua, 2010), steepen the gradient instead of flattening it.

Recent theories have grappled with limitations of early models and have implicated distinct pathways to explain the gradient. For example, articles in differential epidemiology have argued that individual differences, such as personality and cognitive ability, are the "fundamental cause" of the gradient (Gottfredson, 2004),

S. Mason Garrison and Joseph Lee Rodgers, Department of Psychology and Human Development, Vanderbilt University.

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Correspondence concerning this article should be addressed to S. Mason Garrison, Department of Psychology and Human Development, Vanderbilt
AQ: 7 University, ●●●. E-mail: s.mason.garrison@gmail.com

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potentially acting through genetic sources (Arden et al., 2016; Gottfredson, 2004; Marioni, Batty, Hayward, & Kerr, 2014; Marioni et al., 2016). Alternatively, variants of the allostatic load theory (McEwen & Stellar, 1993), such as the Risky Families model (Repetti et al., 2002), implicate the early home-environment. These theory-driven pathways align with interpretations that emerge from behavior genetic models, which decompose relationships into genetic and environmental sources. Surprisingly, little research has applied behavior genetic modeling to understanding the sources of the SES-health gradient. To the best of the authors' knowledge, only one now relatively old article exists that explicitly attempted to do so (Lichtenstein, Harris, Pedersen, & McClearn, 1993, which we discuss in a later section). Other works have treated subissues, such as the relationship between income and psychopathology (South & Krueger, 2011) and between education and physical health (Boardman, Domingue, & Daw, 2015; Johnson et al., 2010), but with measurement or design limitations compared with an overall treatment of SES.

The purpose of this article is not to advocate for a specific fundamental cause of the gradient. Instead, our goal is to narrow the scope of fundamental causal explanations, by explicitly analyzing and untangling the variance sources underlying the gradient using biometrical modeling. Specifically, we will decompose the gradient into genetic (a^2), shared-environmental (c^2), and nonshared environmental (e^2) variance sources, using data from the National Longitudinal Survey of Youth, 1979 (NLSY79).

The SES-Health Gradient and Its Causes

The positive relationship between SES and health (Adler et al., 1994; Antonovsky, 1967; Collins, 1926) is remarkably consistent across place, time, and method of assessment (Oakes & Rossi, 2003; Singh-Manoux, Marmot, & Adler, 2005). For example, the wealthiest 1% of Americans live an average of 10 to 15 years longer than the poorest 1% (Bor, Cohen, & Galea, 2017). College graduates live an average of 8 to 9 years longer than those who did not graduate high school (National Center for Health Statistics, 2012). Clerical (white collar) workers live an average of 8 years longer than unskilled (blue collar) workers (Kaprio, Sarna, Fogelholm, & Koskenvuo, 1996). Conventional explanations of these findings and of the gradient fall into three broad domains: (a) social causation, (b) social selection, and (c) social confounds (Adler et al., 1994; Adler & Ostrove, 1999; Adler & Stewart, 2010; T1 see Table 1 for a summary of these theories).

1. Social causation theories argue that an individual's social standing affects their health (e.g., through allostatic load; Baum et al., 1999; McEwen & Seeman, 1999; McEwen & Stellar, 1993, however, see Matthews, Gallo, & Taylor, 2010).

2. Social selection theories contend that health affects people's abilities to climb the social ladder (e.g., social drift; Fox, 1990).

3. Social confound theories assert that one or more third variables cause health and wealth to covary, instead of either causally influencing the other (e.g., cognitive ability is one such potential confound; see Gottfredson, 2004; Gottfredson & Deary, 2004).

Many theories, such as intergenerational transmission (Haas, 2006) and reciprocal influences (Smith, 1999) involve multiple domains across the life span. Accordingly, multiple pathways could be in play, either simultaneously or sequentially. Or, multiple theories and, thus, different pathways, could simultaneously explain specific health-wealth relationships. Because this topic is so theory-rich, we have tailored our focus to socioeconomic status in adulthood, with some slight discussion of family of origin SES.

Many theorists (e.g., Adler et al., 1994; Adler & Ostrove, 1999; Adler & Stewart, 2010) assert that social causation and selection theories act exclusively through the environment, whereas social confound theories act exclusively through genes. However, these conceptualizations were developed outside of a behavior genetic framework and, thus, lack some of the nuance and insight from current behavior genetics research. We discuss mapping current theory onto behavior genetic modeling in a later section.

Social Causation

Social causation theories vary on two dimensions: baseline (abundance vs. deprivation),[1] and pathway (direct vs. indirect; Fn1 Wagstaff & van Doorslaer, 2000). On the baseline dimension, the baseline outcome (i.e., health) is improved by abundance (e.g., more wealth) or harmed by deprivation (e.g., poverty). Does being poor lead to worse health, or does being rich lead to better health, or both? The pathway dimension illustrates how far apart the cause and effect potentially can be, and how many steps it takes to get from cause to effect (i.e., a very few steps vs. multiple/many steps). Direct pathways connect cause and effect in relatively few steps (typically, one or two; e.g., improved wealth allows greater access to medical care, which improves health; Hummer et al., 1998). Indirect pathways connect cause to effect in multiple/many steps (e.g., poverty leads to living in areas with more stressors, whereby each individual stressor level may or may not reduce health).

Direct models. The Absolute Income Hypothesis (W. Evans, Wolfe, & Adler, 2012; Keynes, 1936) is a direct abundance model, where increased wealth allows individuals to buy better health (e.g., increased wealth may purchase improved access to health care). The equivalent deprivation model is the Absolute Deprivation Hypothesis or Poverty Hypothesis, where decreased wealth costs individuals their health because they are unable to buy health goods (e.g., health insurance). The distinction between these models is subtle and has not been clearly articulated in the literature; abundance models assume that wealth positively influences baseline health, whereas deprivation models assume that lack of wealth negatively influences baseline health.

Variants on these direct models include the Relative Income Hypothesis (Duesenberry, 1967), which suggests that relative increases in wealth allow individuals to buy better health. Similarly, the Relative Status Hypothesis suggests that *relative* increases in status (e.g., wealth, prestige) allow individuals to purchase health. The Relative Status Hypothesis is the broadest of the direct abundance models as it allows SES gains in rank as well as wealth. We direct readers to Lynch et al. (2004) for discussion of the mixed

[1] Wagstaff and van Doorslaer (2000) note that the theoretical distinctions between these dimensions have not be fully articulated in the literature.

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Table 1
Summary of Theories

Broad theory	Summary	Possible biometrical implications
Social causation (Adler et al., 1994; Wagstaff & van Doorslaer, 2000)	An individual's social standing affects their health	(Nonshared-)environmental pathways
Social selection (Haas, 2006)	Health affects people's abilities to climb the social ladder	(Shared-)environmental pathways
Social confounding (Deary et al., 2010; Gottfredson, 2004)	Third variables (e.g., personality, intelligence) influence both social standing and health	Genetic pathways

Note. The biometrical implications are based on Adler and colleagues (Adler et al., 1994; Adler & Ostrove, 1999; Adler & Stewart, 2010), and vary within each broad theory; we slightly revised these interpretations within the text of the current article.

empirical support for direct models, and we note that United States samples show the most support. These direct models have distinct implications for interventions (see, W. Evans et al., 2012, for discussion).

Indirect models. Indirect models of social causation argue that rather than SES directly influencing health, SES places individuals in environments that in turn influence health. In the majority of such models, this indirect influence of health is linked to stress (Adler, 2013), conceptualized both as life events requiring adaptation and as a state where perceived demands exceed their coping abilities. Under allostatic load theory (Baum et al., 1999; McEwen & Stellar, 1993), these environmental stresses of poverty cause "wear and tear on the body" that accumulates over an individual's lifetime. Although longitudinal evidence for this effect in adulthood is mixed (Garrison, Doane, & Elliott, 2018; Matthews et al., 2010), evidence from childhood is not. Specifically, the Risky Families model identifies early childhood environments created by violence, overt aggression, or neglect as sources of poor health across the life span (Repetti et al., 2002). Children of poverty are at greater risk for those household stressors (e.g., Dodge, Pettit, & Bates, 1994; Yoshikawa, Aber, & Beardslee, 2012). Moreover, there is evidence that the effects of poverty on later health are mediated by childhood exposure to early environmental stresses (G. W. Evans & English, 2002).

Social Selection

Social selection theories reverse the causal arrow—instead of SES causing health outcomes, health causes changes in SES. The theories in this area can be partitioned into two mechanisms of action: drift and stunting (Haas, 2006). In drift-based theories, individuals with poorer health gradually drift into lower socioeconomic classes. This downward shift can be the result of discrimination (Ameri et al., 2015), reduced employment (Marwaha & Johnson, 2004), and decreased personal wealth (Chirikos & Nestel, 1985). For example, early longitudinal research on schizophrenia observed between-generation drift and within-generation drift (Goldberg & Morrison, 1963). At their first hospital admission, patients with schizophrenia held similar status jobs to patients with diagnoses of other than schizophrenia (predominantly "anxiety states"). At follow-up (between 1 and 4 years later), patients with schizophrenia had a noticeable within-person decline in job status, whereas patients with other diagnoses had no such decline. In drift models, individuals move down the social ladder.

The other mechanism is stunting, where poor health during critical periods of development negatively impacts an individual's ability to meet key economic or educational milestones. These developmental stages can occur early in life (early childhood; Nelson et al., 2007) or later (e.g., failing to graduate high school; Haynes, 2002). In the longer term, stunting prevents individuals from accumulating status, and thereby inhibits their social mobility.

Lately, these theories have become less popular in the psychology and other behavioral science literature. For example, recent reviews by Adler and Stewart (2010) have explicitly dismissed social selection, whereas other reviews have implicitly dismissed them through omission (e.g., Adler, 2013; Braveman & Gottlieb, 2014) or brief coverage (e.g., W. Evans et al., 2012). Although social selection is often dismissed as the overall cause of the gradient, there likely are subsets of individuals for whom these theories apply. Moreover, the selection effects might be of more importance depending on context, for example, within pretransition underdeveloped countries.

Social Confound

Unlike social causation and social selection theories, social confound theories argue that a third variable causes health and wealth to covary, instead of either causally influencing the other. Predominantly, the third variables identified are individual differences in personality and cognitive ability (Deary, 2010; Deary, Weiss, & Batty, 2010). Although many individual differences are linked with SES and health (Chapman, Roberts, & Duberstein, 2011; Deary et al., 2010), we will focus the discussion on cognitive ability and conscientiousness because their relationships are the most investigated and best understood for both SES and health.

Individual differences and SES. Conscientiousness and cognitive ability are consistently associated with composite measures of SES (Chapman, Fiscella, Kawachi, & Duberstein, 2010; Harwell, Maeda, Bishop, & Xie, 2017; Hernstein & Murray, 1994) and its components (education, occupation, and income; Ng, Eby, Sorensen, & Feldman, 2005; Rodgers et al., 2008; Strenze, 2007). Given that we are interested in these associations as confounds rather than as mediators in causation and selection models, we focus in this review exclusively on how cognitive ability and conscientiousness influence SES. As is the case with many outcomes, cognitive ability and conscientiousness indirectly influence the components of SES through decision-making and various behaviors. These behaviors and decisions are too numerous to review, so we direct readers to the following articles for treatment of the relationships between individual differences in cognition, education, and academic performance (Deary & Johnson, 2010; Kuncel, Ones, & Sackett, 2010; Rowe, Vesterdal, & Rodgers, 1998; Strenze, 2007); occupation and specialization (Judge, Hig-

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gins, Thoresen, & Barrick, 1999; Major, Johnson, & Deary, 2014; Schmidt & Hunter, 2004); income and employment (Behling, 1998; Judge, Martocchio, & Thoresen, 1997; Ng et al., 2005; Strenze, 2007).

Differential epidemiology. Conscientiousness and cognitive ability consistently predict health (Gottfredson, 2004; Hampson, Goldberg, Vogt, & Dubanoski, 2007), and do so across the life-course—including the ultimate measure of health, longevity (Batty, Deary, & Gottfredson, 2007; Jackson, Connolly, Garrison, Leveille, & Connolly, 2015; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007). The effect of cognitive ability on longevity does not vary in strength across most causes of death (Christensen, Mortensen, Christensen, & Osler, 2016). Although health can impact cognitive ability and personality (e.g., traumatic brain injury), we focus exclusively on how these individual differences influence health. Primarily, they do so in two ways. First, cognitive ability and conscientiousness indirectly influence health through decision-making and various health-related behaviors (Bogg & Roberts, 2004; Gottfredson, 2004; Lodi-Smith et al., 2010). Second, cognitive ability is an indicator of overall "system integrity" (Deary, 2012; Lubinski, 2009). System integrity is a "general latent trait of a well-functioning body" (Gale, Batty, Cooper, & Deary, 2009), and reflects how efficiently the human body handles complex systems. "Well-wired" individuals would be more intelligent and healthier on average than "poorly-wired" individuals. This construct is likely related to allostatic load—perhaps well-wired individuals are more able to endure environmental stresses.

Linking Gradient Theories to Environmentality and Heritability

In the previous sections, we have reviewed the three broad classes of theories on causes of the SES-health gradient (i.e., social causation, social selection, and social confounds). Thus far, in previous literature, linkages of theory to biometrical modeling have been sparse. Theorists have asserted that social causation and selection theories act exclusively through the environment, whereas social confound theories act exclusively through genes (Adler et al., 1994; Adler & Stewart, 2010), though we intend to develop and expand these distinctions within this article.

These conceptualizations were developed outside of a behavior genetic framework, and, thus, lack some of the nuance and insight that emerges from the behavior genetics literature. In fact, some of the specific theories can be easily translated into biometrically informed conceptualizations of gene and environmental influences (e.g., the Risky Families Model maps onto the shared-environment; Repetti et al., 2002). Moreover, theories with parsimonious interpretations, such as third variables acting exclusively through genetic pathways, can (and ought to) be broadened to include environmental influences (e.g., Freese & Jao, 2017). Thus, this section will review the biometrically informed conceptualizations of gene and environmental influences.

Environmentality

In behavior genetics, the proportion of variance in a trait that is not because of genes is environmentality. These environmental influences can be divided into shared and nonshared environmental experiences (see Rowe & Plomin, 1981, for early conceptual specification of this distinction). The shared environment, in a biometrical sense, definitionally consists of experiences shared by siblings, whereas the nonshared environment consists of experiences not shared by siblings. These experiences can also be conceptualized as contributing to either between- or within-family variance. The shared environment is exclusively created from between-family experiences. These between-family experiences shared by siblings contribute to family members being more similar to one another than genetic similarity alone would predict. Classic examples of shared-environmental experiences include race, parental discord, divorce, and SES. Shared experiences do not exclusively arrive by means of parents. They can also include sibling shared-experiences (e.g., the shared neighborhood), or even twin-specific experiences (e.g., shared placenta; Neale & Maes, 2004).

The nonshared environment has two sources of nonshared experiences: objective and effective (Goldsmith, 1993; Turkheimer & Waldron, 2000). Objective sources are within-family influences, where explicit differences within a family result in nonshared experiences. These experiences include activities and influences that siblings do not necessarily share such as trips to the museum, educational classrooms, child-specific parenting behaviors, and sibling interactions (see, Rodgers, Rowe, & Li, 1994; Rodgers, Rowe, & May, 1994).

In contrast, "effective" experiences are between-family experiences, in which a shared-event leads to unique experiences. These common experiences include parental discord, divorce, and SES. Early behavior genetic work misattributed these between-family sources of nonshared experiences to the shared environment. Superficially, two children within a certain household will both experience poverty. However, those two hypothetical children experience that same poverty at different stages of development, and in their own idiosyncratic manner, which in turn have the potential to differentially impact them. The majority of nonshared environmental variance appears to come from these between-family experiences because sibling-specific effects only explain a small proportion of environmental variance (Turkheimer & Waldron, 2000).

Heritability

Heritability, the proportion of phenotypic variance because of genes, reflects the extent to which genetic differences contribute to observed individual differences (see Visscher, Hill, & Wray, 2008, for a broad review). Heritability estimates for any construct can vary by age (Bergen, Gardner, & Kendler, 2007), by cohort (Silventoinen, Kaprio, Lahelma, & Koskenvuo, 2000), can be moderated by the environment (e.g., Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003), and do not reflect extreme environments (only those typical within a population). Most, if not all, human behaviors are heritable, but those same behaviors can also have substantial environmental variance as well (most often nonshared; Turkheimer, 2000).

Prior Work

Within Versus Between Family Variance

Some studies have decomposed the SES-health gradient into between- and within-family variance (e.g., Hamdi, South, &

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Krueger, 2016; Monden, 2010; Søndergaard et al., 2012, 2013). However, these studies have used components of SES, rather than the overall construct (as we do in this article, one of the several motivations for the current study). They found sizable between-family variance. In terms of physical health, for example, 20% of education and self-assessed physical health was explained by between-family variance, whereas the remainder was explained by within-family variability (Monden, 2010). Additionally, Søndergaard and colleagues (2012) found that education and all-cause mortality hazard ratios were attenuated by 10–40% when controlling for between-family variance, but still significant, indicating that within-family variance was still meaningful. In terms of mental health, however, between-family variance explained the relationship between education and mental health, completely (see also, Hamdi et al., 2016). Combined, these studies indicate that genetic and/or shared-environmental pathways explain much of the gradient. For physical health, sources of influence originating as between-family variance appear to be a partial explanation, whereas for mental health, between-family variance sources potentially appear to be a complete explanation.

These types of studies tend to examine single components of SES (e.g., education), rather than multiple components (e.g., education and income) or the SES composite of the overall construct (education, income, and occupation). Nevertheless, these findings are suggestive of shared-environmental effects and/or genetic effects. Because this method of separating within- versus between-family variance aggregates shared-environmental and genetic effects, we cannot distinguish between theories that suggest genetic pathways (social confound theories) or shared-environmental pathways (social causation theories).

Biometrical Work

To date, Lichtenstein and colleagues (1993) is the only identified study that decomposes the relationship between multiple components of SES and health into its biometrical sources of variance. They used the Swedish Adoption/Twin Study of Aging (SATSA; 785 twin pairs, including 398 twin-pairs that were raised apart, as well as 387 twin-pairs raised together, and ranging in age from 26 to 86 in 1984). The raised-apart twins were matched with raised-together twins on age, gender, and county of birth. The majority (82%) of raised-apart twins were separated before their 5th birthday. The primary reasons for separation were parental health (e.g., parental illness, parental death) or "economic problems." This cross-sectional analysis used two measures of physical health and multiple components of SES (i.e., material resources, perceived standard of living, education, and occupational status).

They identified a moderate genetic covariance (mean $r_a = .5$) across all components of SES and physical health. The environmental effects were inconsistent. For example, the relationship between education and self-rated health was 27%[2] genetic, 26% shared environmental, and 47% nonshared environmental. Yet, when health was assessed using a chronic illness checklist, there was no genetic influence on its relationship with education. Other work on the biometrical underpinnings of the relationship between education and physical health find consistent genetic linkages (e.g., Boardman et al., 2015; Johnson et al., 2010), as does work on income and psychopathology (e.g., South & Krueger, 2011).

The inconsistency of Lichtenstein and colleagues' results might be because of limitations of their study. They did not create a composite measure of SES, instead relying on the components as separate individual measures. This method has its merits, but it does not allow researchers to broadly evaluate SES as a cause of the gradient. Further, the authors did not address potential selection effects caused by health- or wealth-related twin separations, or survivor effects, given the age of the subjects. Regardless, their results were a promising first step into identifying the sources of the gradient and have helped motivate the current study.

Current Study

To summarize, the current study examines the relationship between SES and health, using biometrical modeling and data from a national household probability sample, the NLSY79. Health was measured as respondents passed age 40, using the physical health component summary score (PCS) of the 12-Item Short Form Health Survey (SF-12; Ware, Kosinski, & Keller, 1995), and the mental health component summary score of the SF-12 (MCS).

This examination extends the SES-health gradient literature in several key ways. First, we decompose the relationship between SES and health into genetic and environmental components, using a national probability sample and, thus, have findings that are approximately representative across levels of SES. Second, we use a dataset with kinship pairs occurring at a level approximately representative of those in U.S. families, which naturally improves external validity compared with traditional twin or adoption designs. Third, we distinguish between mental and physical health in these analyses, and provide analytic results from each domain separately, as well as a combined analysis of both domains together. Finally, we evaluate various theories within the context of these results, identifying ones that are inconsistent with our empirical findings.

The results from Lichtenstein and colleagues (1993) are our primary basis for making predictions. However, as noted above, these results are from a relatively old and data-limited sample. In general, they supported a genetic component and a small shared-environmental component influencing the bivariate relationship between SES and physical health. Additional literature on the relationship between education and health is consistent, also supporting a genetic effect (Johnson et al., 2010). Lichtenstein and colleagues (1993) provided no guidance for mental health. Other biometrical literature provides some guidance based on between- versus within-family modeling, suggestive of either genetic or shared-environmental effects, or both (e.g., Hamdi et al., 2016).

First, we will conduct univariate biometrical analyses on SES, physical health, and mental health. The best fitting models will be determined by a series of nested model comparisons. The univariate results from the best fitting models will guide model fitting for later stages. To minimize bias in the final model, we elected to be conservative with regard to excluding model parameters. We would prefer to leave an unnecessary parameter in the model, reducing precision, rather than to exclude a parameter that belongs, inducing bias.

[2] Percentages were derived from Figure 4 in Lichtenstein and colleagues (1993).

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Second, we will conduct bivariate biometrical analyses on SES with physical health, and SES with mental health. These models will be guided by the univariate results. Specifically, all tests of whether a correlation has a heritable component are contingent on both variables having heritable components at the univariate level. For example, if SES does not have a heritable component, then there can be no common shared heritable component between SES and any measure of health. This same logic applies to shared-environmental components. Again, the best fitting models will be determined by series of nested model comparisons.

Finally, a trivariate model will be estimated, linking SES, physical health, and mental health. The best fitting bivariate models will be merged to create the trivariate model. Again, the best fitting model will be determined by a series of nested model comparisons.

The NLSY79 data were collected by professional survey organizations, and are publicly available online. Ethics approval was granted by the institutional review boards of the Ohio State University, the University of Chicago's National Opinion Research Center, and U.S. Office of Management and Budget. Additionally, Vanderbilt University's Institutional Review Board approved the use of the NLSY data in our lab, and classified it as exempt, because of the public access and de-identified status of our archival data.

Method

Models

Specification, estimation, and missing data. All models were specified using Mplus (Version 7.4; Muthén & Muthén, 2017), and estimated using full information maximum likelihood (FIML) to account for missing data. The univariate ACE model specification is illustrated with a simplified path diagram in Figure 1. We note that the model is not saturated because the correlation between a_1 and a_2 varies by level of relatedness. The bivariate correlated factors ACE model specification is illustrated with the path diagram in Figure 2. The diagram is simplified to only include one member of the kin pair; an identical model exists for the second member of the kin pair (subscripted with 2, as in Figure 1), and the latent genetic and shared environmental components are linked as in Figure 1. Because full-siblings, half-siblings, and cousins differ in their level of relatedness, the model is not saturated. Sample Mplus syntax, adapted from Prescott (2004), is provided in the online supplemental materials.

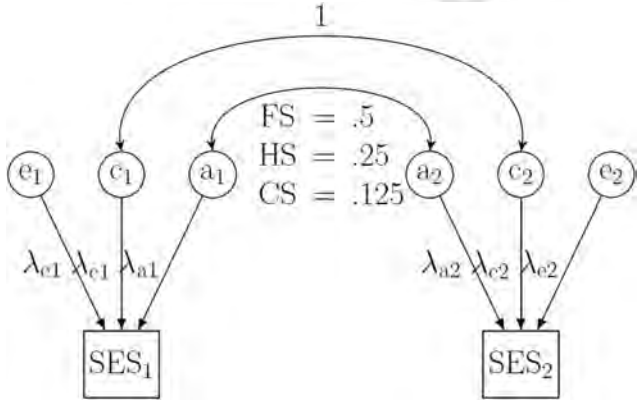

Figure 1. Path diagram illustrating model specification for a univariate ACE model. a = additive genetic factor; λ_a = additive genetic loading; c = shared-environmental factor; λ_c = shared-environmental loading; e = nonshared-environmental factor; λ_e = nonshared-environmental loading; $SES_{1,2}$ = socioeconomic status measure for kin 1 and kin 2; FS = full siblings; HS = half siblings; CS = conditional stimulus, cousins.

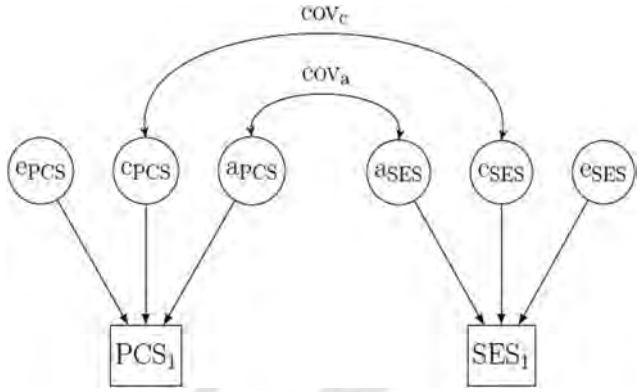

Figure 2. Path diagram illustrating model specification for a bivariate correlated factors ACE model. The diagram is simplified to only include one member of the kin pair. a = additive genetic factor; λ_a = additive genetic loading; cov_a = covariance between additive genetic factors; c = shared-environmental factor; λ_c = shared-environmental loading; cov_c = covariance between shared-environmental factors; e = nonshared-environmental factor; λ_e = nonshared-environmental loading.

Terminology. Throughout this article, we refer to the covariance between genetic factors (cov_a), the genetic correlation (r_a), and the bivariate heritability These terms are related, but not identical constructs. The covariance between genetic factors describes the degree of overlap between the genetic variance in physical health with the genetic variance in SES. The genetic correlation reflects the correlation between physical health and SES that is attributable to the covariance between genetic factors. The bivariate heritability describes the proportion of the (phenotypic) correlation between physical health and SES that is attributable to the covariance between genetic factors.

Similarly, we refer to the covariance between shared-environmental factors (cov_c), the shared-environmental correlation (r_c), and the bivariate environmentality. Again, these terms are related, but not identical. The covariance between shared-environmental factors describes the degree of overlap between the shared-environmental variance in mental health with the shared-environmental variance in SES. The shared-environmental correlation reflects the correlation between mental health and SES that is attributable to the covariance between shared-environmental factors. The bivariate environmentality is the proportion of (phenotypic) correlation between mental health and SES that is attributable to the covariance between shared-environmental factors.

Subject characteristics. The NLSY79 dataset (see Garrison & Rodgers, 2016 for detailed description), is based on a nationally representative household probability sample. On December 31, 1978, 12,686 adolescents were sampled within a household probability sample of 8,770 households. The initial sample consisted of three subsamples (two civilian and one military); the civilian

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subsamples were a cross-sectional household probability sample of 6,111 noninstitutionalized adolescents residing in the United States on December 31st of 1978; and a separate oversampled civilian subsample of 5,295 racial minority and disadvantaged white respondents. Subjects were between the ages of 14 and 21 on the sampling date. Participants were surveyed annually until 1994, and then surveyed biennially to the present. Information on the sampling process, attrition, and access to the data can be found on the US Bureau of Labor Statistics (BLS) Web site (http://www.bls.gov/nls/nlsy79.htm). A bibliography of articles using these publicly available data can be found at https://nlsinfo.org/bibliography-start. Of the nearly 6,000 papers listed using the NLSY79, we found no cases that examined the biometrical relationship between SES and health.

To conduct this study using the requisite within-family information, we used kinship pairs to support biometrical analysis. Our research team has recently completed a multiyear project to reliably and validly identify the NLSY79 kinship pairs (Rodgers et al., 2016) using both indirect and direct ascertainment of relatedness. Full-sibling (n = 4,006 pairs), half-sibling (n = 297 pairs), and cousins (n = 96 pairs, who lived together in the same household in 1979) are used in the current study. Sample sizes for each level of kinship-relatedness varied because they are approximately representative of the distribution of kinship pairs in the population of households at the time the NLSY79 sample was defined. Other research teams who wish to use the NLSY kinship links—from the NLSY79 used here, or kinship links from the NLSY-Children/Young Adults or the NLSY97 data—will find those online at http://liveoak.github.io/NlsyLinks/. This Web site includes reproducible examples in R and SAS, as well as user support services. These kinship pairs can be used for behavior genetic modeling (e.g., Boutwell et al., 2017; Rodgers et al., 2015), sibling comparison designs (Garrison & Rodgers, 2016; Hadd & Rodgers, 2017), and other methods of causal inference.

Health Measures

The youngest NLSY79 respondents passed age 40 in 2005 (and then were surveyed in 2006). As they passed age 40, the NLSY79 respondents completed a module of health questions, including the 12-Item Short Form Health Survey (SF-12; Ware et al., 1995). The SF-12 was designed to assess health functioning and overall health-related quality of life. It contains two subscales measuring those general qualities for physical health (physical component summary score; PCS) and for mental health (mental component summary score; MCS). Both subscales correlate highly ($r > .9$ with their respective longer-forms versions Gandhi et al., 2001),and have moderately high test–retest reliability (α_{pcs} = .89; α_{mcs} = .76; Ware et al., 1995).

Administration of the health survey module began in 1998, when the oldest respondents (i.e., those born in 1957 and 1958) reached age 40. The module continued to be administered biennially until the youngest respondents reached age 40 and were interviewed in 2006. Approximately half of subjects took the Health 40 module at age 40 and half at 41, because of the biennial schedule of NLSY79 survey administration. More details about these health modules are provided in the subsections below. Additional sample items and psychometric properties can be found here: https://www.nlsinfo.org/content/cohorts/nlsy79/topical-guide/health.

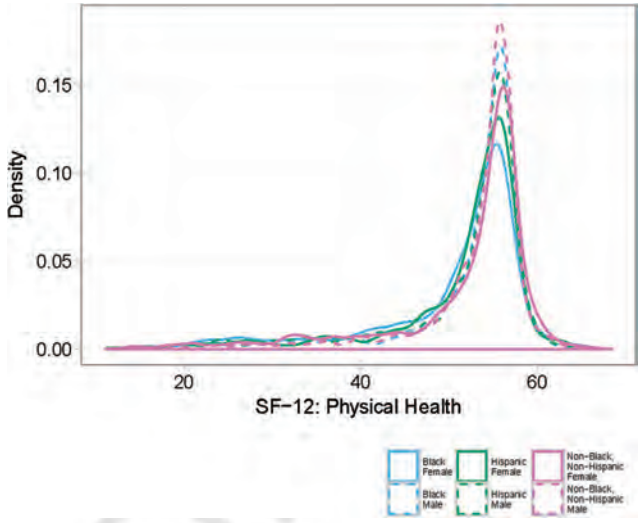

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Figure 3. Density plot of 12-Item Short Form Health Survey (SF-12): Physical health at age 40 by sex and race. See the online article for the color version of this figure.

Physical health (PCS). Higher scores on the physical component summary scores of the SF-12 (PCS) indicate greater physical health functioning/quality. Sample items include[3]: "In general, would you say your health is?"; "During the PAST 4 WEEKS have you had any of the following problems with your work or other regular activities AS A RESULT OF YOUR PHYSICAL HEALTH?". The scores varied by sex and race.[4] These differences were significant, but not large in magnitude. Mean scores for women (51.4, SD = 8.7) were significantly lower than mean scores for men (52.6, SD = 7.3); $p < .001$. Black respondents (51.3, SD = 8.4) and Hispanic respondents (51.8, SD = 8.1) had significantly lower mean scores ($p < .001$) than non-Black, non-Hispanic respondents (52.5, SD = 7.8). Throughout this article, race has been dichotomized into a minority status variable. These two groups consist of Non-Black, Non-Hispanic respondents (coded 0); and Black and/or Hispanic respondents (coded 1). Descriptive statistics used the three-category variable denoted as race, whereas biometrical models used the two-category variable denoted as minority status. Figure 3 characterizes the distribution of PCS, grouped by sex and race with smoothed density plots.

Mental health (MCS). Higher scores on the mental health component summary score of the SF-12 (MCS) correspond with greater mental health functioning/quality. Sample items include: "Have you felt downhearted and blue?"; "During the PAST 4 WEEKS, were you limited in the kind of work you do or other regular activities AS A RESULT OF ANY EMOTIONAL PROBLEMS (such as feeling depressed or anxious)?" MCS scores varied by sex, but not race. Figure 4 characterizes the distribution of MCS, grouped by sex and race with smoothed density plots. Mean scores for women (51.9, SD = 8.9) were significantly lower than

[3] Capitalization is from the original.

[4] Race was defined by the original NLSY79 investigators based on a combination of self-identification, interviewer report, and inference from household reports (Ward & Burich, 1978). This method resulted in three groups: Hispanic, Black, and non-Black-non-Hispanic respondents.

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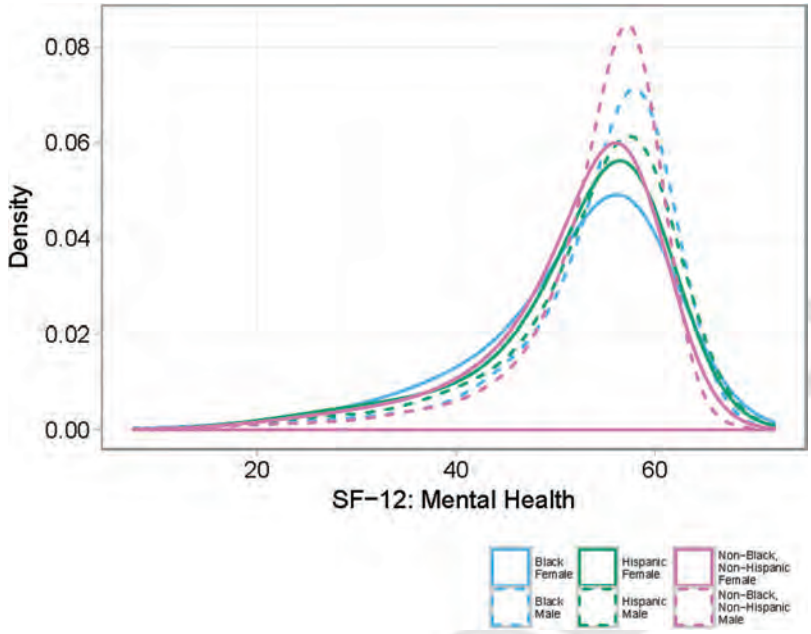

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Figure 4. Density plot of 12-Item Short Form Health Survey (SF-12): Mental health at age 40 by sex and race. See the online article for the color version of this figure.

mean scores for men (54.0, *SD* = 7.7; $p < .001$). Black respondents' mean scores (53.0, *SD* = 8.7) and Hispanic respondents (52.9, *SD* = 8.7) did not differ significantly ($p = .86$) from non-Black-non-Hispanic respondents' mean (52.9, *SD* = 8.0). Although sex differences were significant ($p < .001$), those differences were small.

Normality. The distributions of both PCS and MCS were highly negatively skewed, that is, they were not normally distributed; see the normal QQ plots in Figure 5. We transformed the SF-12 measures using Box-Cox transformations (Box & Cox, 1964).[5] The transformed variables' normal QQ plots are also displayed in Figure 5, and were much closer to normal. Analyses using the untransformed variables gave very similar heritability estimates, albeit with poorer model fits.

Heritability from past studies. There is limited information on the heritability of the SF-12. The Danish Twin Registry collected PCS and MCS from the SF-12 (Johnson et al., 2010; Steenstrup, Pedersen, Hjelmborg, Skytthe, & Kyvik, 2013) at mean age 45 (*SD* = 13.7). PCS and MCS distributions were skewed in the same manner as our sample (Johnson et al., 2010; Steenstrup et al., 2013). Median values for PCS (55.9) and MCS (54.4) were comparable with nonminorities in the NLSY79 sample ($PCS_{median} = 55.3$, $MCS_{median} = 55.6$). Neither Johnson and colleagues (2010) nor Steenstrup and colleagues (2013) reported univariate ACE estimates. However, Johnson and colleagues (2010, Figure 3) illustrated that the variance components for PCS were moderated by educational attainment. At a 7th grade education, a^2 was 0.63 for adult men (0.76 for adult women), c^2 was 0.02 (0.02), and e^2 was 0.36 (0.22). At high school education, a^2 was 0.43 for men (0.58 for women), c^2 was 0.04 (0.03), and e^2 was 0.53 (0.39). At more than 4 years of education beyond high school, a^2 was 0.15 for men (0.23 for women), c^2 was 0.07 (0.05), and e^2 was 0.78 (0.71).

Steenstrup and colleagues (2013) provided enough information to derive univariate values, using Falconer's (Falconer, 1952) formula. The following calculations are for subjects between ages 35–54. For MCS, a^2 was 0.17 for men (0.31 for women), c^2 was 0.09 for men (−0.01), and e^2 was 0.73 for men (0.69). Given that the samples were derived from Nordic populations and we are unable to provide confidence intervals for these calculations, we consider these findings to be only partially informative for comparison to our U.S. sample—at least slightly suggestive of an AE model for physical health (PCS) and an ACE model for mental health (MCS).

Socioeconomic Status

We constructed an SES measure from the NLSY79 based on Myrianthopoulos and French (1968) and used more recently by Turkheimer and colleagues (2003). Each subject was given an aggregate score based on the mean of their total-net-family income, education, and occupation quantile scores. Subjects with missing data were not excluded—instead, their aggregated scores were created from their nonmissing components. Higher scores correspond to higher SES.

SES was computed for the same year as the Health 40 module (*M* = 52.1, *SD* = 21.8). The distribution of this index by race and sex is illustrated in Figure 6. Average scores for women (52.8, *SD* = 21.9) were significantly higher than average scores

[5] We used the powerTransform function from the car package (Fox & Weisberg, 2010), which uses a maximum-likelihood adaptation of Box-Cox to estimate a transformation that normalizes a distribution.

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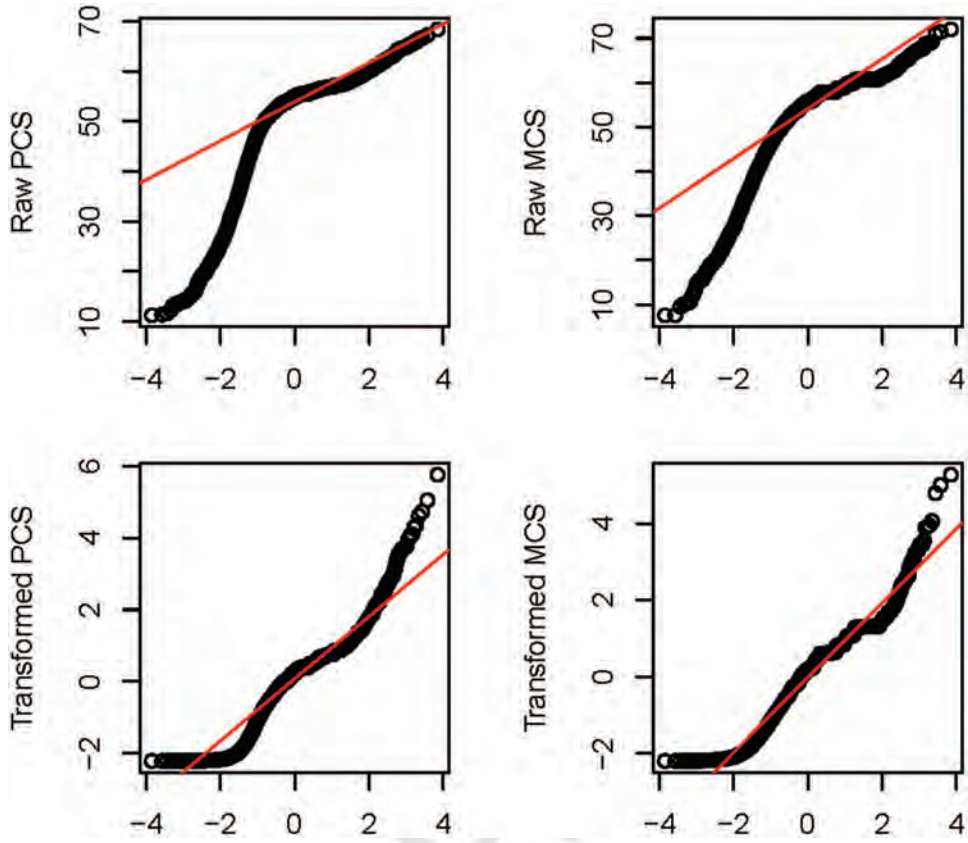

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Figure 5. Normal QQ plots of untransformed and transformed physical health component summary score (PCS) and mental health component summary score (MCS). See the online article for the color version of this figure.

AQ: 8

Fn6 for men (51.3, $SD = 21.6$); $t(7322.04)^6 = 2.87$, $p < .001$. Black respondent mean scores (47.9, $SD = 21.7$) and Hispanic respondent mean scores (48.2, $SD = 21.6$) were significantly lower than the mean for non-Black-non-Hispanic respondents (56.1, $SD = 21.1$), $t(7325.12) = -16.07$, $p < .001$.

Heritability from past studies. We found no instances of Myrianthopoulos and French's (1968) system for coding SES decomposed into its biometric components. However, recent reviews in economics provide evidence for both genetic ($a^2 \approx .5$) and shared-environment effects ($c^2 \approx .1$) for the components commonly used in SES composites (Benjamin et al., 2012), as do findings in psychology (Plomin & Bergeman, 1991; Rowe et al., 1998). Moreover, Lichtenstein and colleagues (1993) found evidence for meaningful genetic and environmental variance for their multiple components of SES.

Results

The observed correlation between SES and the rescaled PCS was .2; the observed correlation between SES and the rescaled MCS was .08. Table 2 displays the correlation matrix between these variables at age 40. The diagonal indicates the sample size for each variable, and the upper triangle shows the number of respondents with viable scores for both respective variables. Because of the data missingness, all models were estimated using full information maximum likelihood (FIML). FIML was restricted to subjects with the same levels of relatedness. Additionally, all models allowed means to vary across kinship groups.

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Univariate Results

Table 3 reports the estimated variance components and model fit statistics for univariate ACE models of SES, PCS, and MCS, controlling for sex and minority status at age 40. To maximize power for a final trivariate model, we are conservative in regard to excluding variance components at the univariate (and bivariate) level.

T3

Socioeconomic Status. The best fitting model for the heritability of SES, controlling for minority status and sex, was an ACE model. In general, model fit statistics were excellent. The χ^2 test of the overall model was not significant ($p = .07$), indicating good model fit. Root mean square error of approximation (RMSEA) indicated that the model fit closely (RMSEA = .02, 90% confidence interval (90% CI [0, .03]) and that perfect fit could not be rejected. Standardized root mean square residual (SRMR) = .02, indicating good model fit. Because we were prioritizing maximum power and minimal bias in the final model, we retained the shared-environmental parameter.

[6] *t* tests were conducted without assuming equal variances by adjusting degrees of freedom with the Welch-Satterthwaite formula.

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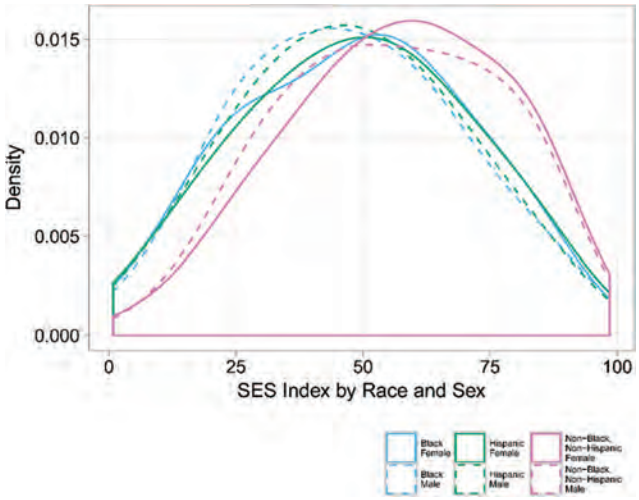

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Figure 6. Density plots of socioeconomic status (SES) by race and sex. See the online article for the color version of this figure.

Minority status and sex accounted for 5% of the total variance in SES. After adjusting for those covariates, $a^2 = 57\%$ ($p < .001$; 90% CI[7] [.27, .98]), $c^2 = 8\%$ ($p = .08$; 90% CI [.002, .35]), and the nonshared environment accounted for the remainder, $e^2 = 35\%$ ($p < .001$; 90% CI [.19, .57]). Fn7

Physical health (PCS). The best fitting model for the heritability of PCS was an AE model. It was statistically indistinguishable from an ACE model ($\chi^2 = 0$, $p = 1.00$) because the c^2 estimate was zero in both models. Model fit statistics were good. Controlling for covariates in the AE model, the χ^2 test of model fit ($p = .09$) was not significant, indicating good model fit. RMSEA indicated that the model fit closely (RMSEA = .02, 90% CI [0, .03]) and could not reject perfect fit; SRMR = .02 was low.

Minority status and sex accounted for 2% of the total variance in PCS. After adjusting for those covariates, $a^2 = 17\%$ ($p < .001$; 90% CI [.11, .23]), whereas the nonshared environment accounted for the remainder, $e^2 = 83\%$ ($p < .001$; 90% CI [.77, .9]).

Mental health (MCS). The best fitting model for MCS was a CE model. It was statistically indistinguishable from an ACE model ($\chi^2 = 0$, $p = 1.00$) because the a^2 estimate was zero in both models. The χ^2 test was not significant ($p = .8$), indicating good model fit. RMSEA indicated that the model fit closely (RMSEA = 0, 90% CI [0, .01]) and that perfect fit could not be rejected. SRMR = .02, also indicating good model fit.

Minority status and sex accounted for 2% of the total variance in MCS. After adjusting for those covariates, the shared environment (c^2) accounted for 9% ($p < .001$; 90% CI [.06, .12]) of the variance in MCS, whereas the nonshared environment accounted for the remainder, $e^2 = 91\%$ ($p < .001$; 90% CI [.88, .94]).

Table 2
Correlation and Sample Sizes

	SES	PCS	MCS
SES	8,465	8,402	8,402
PCS	.198*	8,402	8,402
MCS	.083*	−.006	8,402

Note. SES = socioeconomic status; PCS = physical health component summary score; MCS = mental health component summary score.
* Indicates significance, $p < .001$.

Table 3
Estimates and Fit Statistics for Best Fitting Univariate Models

	ACE SES	AE PCS	CE MCS
a^2	.57	.17	
c^2	.08		.09
e^2	.35	.83	.91
Proportion of variance attributable to covariates	.05	.02	.02
$p(\chi^2\ M, df \approx 25)$	.07	.09	.8
CFI	.99	.92	1.00
TLI	.99	.94	1.00
RMSEA (90% CI)	.02 [0, .03]	.02 [0, .03]	0 [0, .01]
SRMR	.02	.02	.02

Note. SES = socioeconomic status; PCS = physical health component summary score; MCS = mental health component summary score; CFI = comparative fit index; TLI = Tucker Lewis Index; RMSEA = root mean square error of approximation; CI = confidence interval; SRMR = standardized root mean square residual.

Bivariate Results

Both meaningful genetic and shared-environmental variance exists in the measure of SES. The question remains whether the genetic variance in PCS and the shared-environmental variance in MCS overlap with the equivalent variance source in SES. Thus, a series of correlated-factors models were run to identify the best fitting models of the relationship between SES and a single measure of health. All models controlled for minority status and sex.

Bivariate models of SES and physical health (PCS). A nested model comparison found that allowing the genetic effects to covary improved the fit of the model dramatically; $p(\chi^2) < .001$. Model fit statistics are provided in Table 4 and were generally good. Although the χ^2 test of model fit was significant ($p < .001$), RMSEA (.03, 90% CI [.03, .04]) indicated close fit and SRMR = .04 was low. Standardized model parameter estimates are displayed in Figure 7 with *SE*s in parentheses, adjusted for minority status and sex. T4 F7

After adjusting for covariates, the estimated parameters for SES were $a^2 = 55\%$ ($p < .001$; 90% CI [.26, .95]), $c^2 = 6\%$ ($p = .13$; 90% CI [0, .35]), $e^2 = 39\%$ ($p < .001$; 90% CI [.23, .59]). The estimated parameters for PCS were $a^2 = 10\%$ ($p < .001$; 90% CI [.05, .17]) and $e^2 = 90\%$ ($p < .001$; 90% CI [.84, .96]). The bivariate estimates of the separate components were nearly identical to the univariate estimates. Again, we elected to keep the shared-environmental variance component of SES in the model to minimize bias in the final model.

The estimated covariance between the genetic components of SES and PCS was .62 ($p < .001$); 95% CI [.28, .97], which means that 62% of the genetic influence was common to both measures. We calculated the genetic correlation ($\hat{r}_a$) between SES and PCS with path tracing rules (Wright, 1934):

[7] Alpha was set to .05 throughout this article. We provided 90% confidence intervals of squared parameter estimates for readers interested in testing unidirectional hypotheses.

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Table 4
Bivariate and Trivariate Model Fits

	PCS and SES	MCS and SES	PCS, MCS, and SES
$\chi^2(df)$	151 (62)	65.6 (62)	197 (111)
CFI	.92	.996	.94
TLI	.93	.997	.94
RMSEA (90% CI)	.03 [.03, .04]	.01 [0, .02]	.02 [.02, .03]
p(RMSEA) $<$ .05	1	1	1
SRMR	.04	.02	.035

Note. SES = socioeconomic status; PCS = physical health component summary score; MCS = mental health component summary score; CFI = comparative fit index; TLI = Tucker Lewis Index; RMSEA = root mean square error of approximation; CI = confidence interval; SRMR = standardized root mean square residual.

$$\hat{r}_a = \beta_{PCS,a_{pcs}} * \Psi_{PCS,a_{SES}} * \beta_{SES,a_{SES}}$$

$$\hat{r}_a = 0.32 * 0.62 * 0.74 = 0.15$$

The observed genetic correlation ($\hat{r}_a$) was .15, 95% CI [.12, .16]. Consequently, the bivariate heritability was .91 (95% CI [.72, .97]). As a more conservative test, we substituted the latent phenotypic correlation (r = .16) with the observed correlation between SES and PCS (r = .2); in this case, the bivariate heritability was .75 (95% CI [.59, .8]). Both bivariate heritabilities indicated that a large component of the gradient was heritable, and the remaining phenotypic variance was explained by the nonshared environment.

Bivariate models of SES and mental health (MCS). A nested model comparison found that allowing the shared-environmental effects to covary improved the fit of the model dramatically, $p(\chi^2) < .001$. Model fit statistics are provided in Table 4 and were generally excellent. The χ^2 test of model fit was not significant (p = .35), and RMSEA (.01, 90% CI [0, .02]) could not reject perfect fit. SRMR = .02 was low. Standardized and covariate-adjusted model parameter estimates are displayed in Figure 8 with *SE*s in parentheses.

After adjusting for minority status and sex, the estimated parameters for SES were a^2 = 52% ($p < .001$; 90% CI [.22, .93]), c^2 = 10% (p = .03; 90% CI [.001, .35]), e^2 = 39% ($p < .001$; 90% CI [.22, .59]). The estimated parameters for MCS were c^2 = 8% ($p < .001$; 90% CI [.06, .12]) and e^2 = 92% ($p < .001$; 90% CI [.89, .95]). The bivariate estimates of the separate components were nearly identical to the univariate estimates.

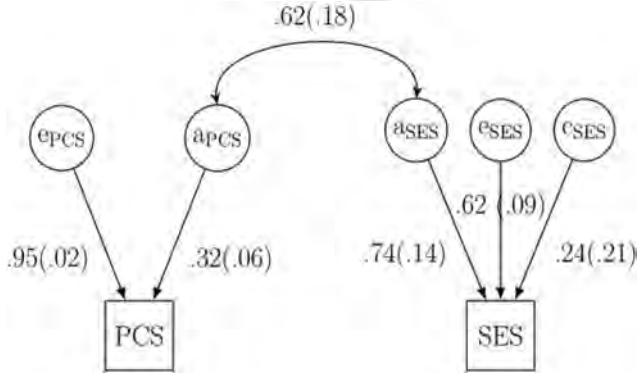

Figure 7. Bivariate correlated factors model of socioeconomic status (SES) and physical health component summary score (PCS).

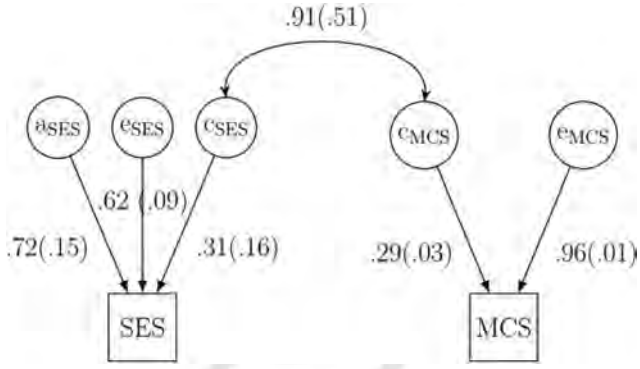

Figure 8. Bivariate correlated factors of socioeconomic status (SES) and mental health component summary score (MCS).

The covariance between the shared-environmental components of SES and MCS was .91 (p = .08; 95% CI [0, 1]), which means that 90.9% of the shared-environmental influences was common to both measures. We calculated the shared-environmental correlation ($\hat{r}_c$) between SES and MCS with path tracing rules (Wright, 1934).

The shared-environmental correlation $\hat{r}_c$ = .08 (95% CI [.06, .1]). Consequently, the bivariate environmentality was 1 (95% CI [.94, 1]). As a more conservative test, we substituted the latent phenotypic (r_c = .06) correlation with the observed correlation between SES and MCS (r = .08); this bivariate environmentality was .986 (95% CI [.7, 1]).

Trivariate Results

Combining the bivariate models, we examined the simultaneous influence of SES on mental and physical health. A series of nested model comparisons found the following:

- Allowing the shared-environmental effects between mental health and SES to covary improved the fit of the model ($p(\chi^2) < .001$);
- Allowing the genetic effects between physical health and SES to covary improved the fit of the model ($p(\chi^2) < .001$);
- Allowing the genetic effects between physical health and SES to covary after already covarying the shared-environmental effects between mental health and SES improved the fit of the model ($p(\chi^2) < .001$); and
- Allowing the shared-environmental effects between mental health and SES to covary after already covarying the genetic-environmental effects between physical health and SES improved the fit of the model ($p(\chi^2) < .001$).

Model fit statistics were excellent (see Table 4). Although the χ^2 test of model fit ($p < .001$) was significant, RMSEA (.02, 90% CI [.02, .03]) indicated close fit; SRMR = .04 was low. Estimates were very similar in the trivariate analysis to those from the separate bivariate analyses.

After adjusting for minority status and sex, the estimated parameters for SES were a^2 = 49% ($p < .001$; 90% CI [.39, 61]),

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c^2 = 8% ($p < .001$; 90% CI [.04, .14]), e^2 = 42% ($p < .001$; 90% CI [.35, .50]). The estimated parameters for PCS were a^2 = 10% ($p < .001$; 90% CI [.05, .17]) and e^2 = 90% ($p < .001$; 90% CI [.84, .96]). The estimated parameters for MCS were c^2 = 8% ($p < .001$; 90% CI [.06, .12]) and e^2 = 92% ($p < .001$; 90% CI [.89, .94]). Standardized and covariate-adjusted model parameter estimates are displayed in Figure 9 with *SE*s in parentheses.

The covariance between the genetic components of SES and PCS was .65 ($p < .001$; 95% CI [.37, .94]). We calculated the genetic correlation ($\hat{r}_a$) between SES and PCS with path tracing rules (Wright, 1934). The genetic correlation ($\hat{r}_a$) was .15 (95% CI [.12, .16]). Consequently, the bivariate heritability was .91 (95% CI [.72, .98]). As a more conservative test, we substituted the latent phenotypic correlation (r = .16) with the observed correlation between SES and PCS (r = .2); the bivariate heritability .75 (95% CI [.59, .81]). Both heritabilities indicated that a large component of the gradient was heritable, and the remaining covariance was explained by the nonshared environment.

The covariance between the shared-environmental components of SES and MCS was .999 ($p < .001$; 95% CI [.89, 1]). The shared-environmental correlation ($\hat{r}_c$) was .08 (95% CI [.06, .1]). Consequently, the bivariate environmentality was 1 (95% CI [.94, 1]). As a more conservative test, we substituted the latent phenotypic correlation (r_c = .06) with the observed correlation between SES and MCS (r = .08); this bivariate environmentality was .96 (95% CI [.71, 1]).

Internal and External Validity

We conducted sensitivity analyses to evaluate internal and external validity of our findings. As one approach to evaluate internal validity, we replicated all our analyses presented in this article with the inclusion of monozygotic (MZ) twin pairs (there were a total of 11 MZ twin pairs in the NLSY79 sample). Although the addition of MZ twins decreased model fit, fit statistics were generally good. Regardless, parameter estimates across models were practically identical, primarily differing at the second decimal place.

In addition, we replicated all analyses presented in this article by replacing the Mental Health Component with CES-D. Although CES-D had a larger correlation with SES (r = .2), CES-D was highly skewed and bimodal, even after Box-Cox transformation. In those analyses, all results replicated, including the lack of genetic effect. However, those models did not fit as well, likely because of the skewed and bimodal nature of CES-D.

To evaluate external validity, we replicated the final trivariate model using the NLSY79 National Probability Subsample (n = 6,111 noninstitutionalized adolescents), which consists of the cross-sectional household probability subsample described earlier. Again, fit statistics were generally excellent. Parameter estimates were similar, with considerable overlap between their 95% CIs. Moreover, bivariate heritabilities and environmentalities were also similar and had considerable confidence interval overlap with our original results. For physical health, the latent bivariate heritability was 0.86, 95% CI [0.67, 0.97] (observed correlation 0.74, 95% CI [0.57, 0.83]). For mental health, the latent bivariate environmentality was 1, 95% CI [0.72,1] (observed correlation 0.94, 95% CI [0.58, 1]). The similarity between the original results and the results from the sensitivity analyses support the internal and external validity of our findings.

Discussion

This article presents a biometrical decomposition of the bivariate relationship between SES and Health—often called the SES-health gradient—for both mental and physical health at age 40. The identified general pathways of influence of the SES-health gradient differ by aspect of health. SES links to mental health through shared-environmental (and nonshared environmental) pathways. In contrast, SES links to physical health through genetic (and nonshared environmental) pathways.

For physical health, our results are consistent with the Lichtenstein and colleagues (1993) finding of a genetic pathway linking physical health and various components of SES. In our study, the genetic pathway explained the bulk of the gradient, even using the most conservative bivariate heritability we could devise by substituting the latent phenotypic correlation with the observed correlation. For mental health, our findings diverged from those for physical health. We observed no meaningful genetic effect and instead found the shared-environment explained practically the entire mental health gradient at age 40. Again, this relationship held even when using the most conservative bivariate environmentality we could devise. These results suggest that Adler (Adler et al., 1994; Adler & Stewart, 2010) and Gottfredson (2004) are both

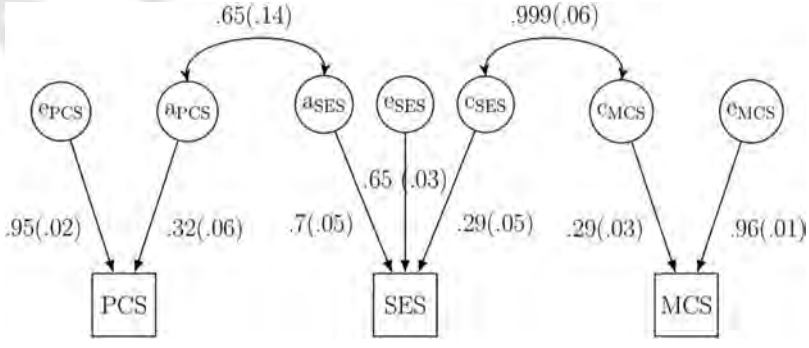

Figure 9. Trivariate correlated-factors model of socioeconomic status (SES), physical health, and mental health at age 40.

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correct—the environment matters, genes matter, but the nature of the health outcome is critical.

Physical Health

Across models, genetic variance underlies the gradient for physical health. This percentage ranged from 70% genetic and 30% nonshared environment in conservative calculations to 90% genetic and 10% nonshared environment in more traditional calculations. Regardless of the method of calculation, this finding was comparable to those of Lichtenstein's Nordic sample (1993; 67% genetic)—even though they used a different type of sample, twins raised apart—that lends support to this result. Of course, this finding does not mean the gradient is fixed or cannot respond to intervention. On the contrary, the high bivariate heritability simply identifies the source of overlap between SES and health as emergent from biological sources. Both gene-environment interactions and epigenetic sources provide many explanations for why such genetic overlap does not imply deterministic outcomes.

Mental Health

Across models, the shared-environment was the primary source of the gradient for mental health at age 40. This percentage ranged from 90% shared-environment and 10% nonshared environment in conservative calculations to 100% shared environmental in more traditional calculations. The results consistently indicated that the shared-environment explained practically all of the small correlation between SES and mental health, and were replicated using a depressive symptomology measure. Such findings suggest that early experiences in the home may be important influences on the mental health aspect of the gradient, and are consistent with indirect models of social causation (and selection). Because the mental health findings are derived from small shared-environmental effects ($c^2 < .10$), the effects were relatively less robust than the equivalent genetic effects for physical health (e.g., wider confidence interval). Accordingly, high-powered research is needed to cross-validate these effects.

Theoretical Implications

Theorists (notably, Adler and various colleagues; Adler et al., 1994, Adler & Stewart, 2010) assert that social causation and selection theories act exclusively through the environment, whereas social confound theories act exclusively through genes. Using their framework, the genetic effect of physical health with SES provides definitive support for social confound theories and all but eliminates social causation (and selection) theories; whereas the shared-environmental effect underlying mental health with SES maps onto social causation (and selection) theories, accordingly eliminating social confound explanations. Such an interpretation may be overly simplistic, however.

The relationship between SES and health is dynamic and temporal—in other words, complex. For example, schizophrenia is often used as an illustration of selection (Goldberg & Morrison, 1963). Accordingly, theories of selection would predict that schizophrenia is not heritable and predominantly explained by environmental sources. Yet, schizophrenia is 64 to 89% heritable (National Institute of Mental Health's Genetics Workgroup, 1998; Tsuang, 2000). How can SES-health gradient theory and behavior genetics appear incompatible? They are not. The estimates given by behavior genetics refer to distal causes, whereas the gradient theories refer to more proximal causes.

Instead, we ought to think of these two pieces as giving predictions at different points in the causal stream. The proximal causes are conditioned on distal causes. For example, given an individual genetically predisposed to developing schizophrenia, social selection theories predict the following chain of events:

- The at-risk individual experiences schizophrenia, then
- A downward drift into poverty occurs.

Social causation theories predict an alternative chain of events:

- The at-risk individual experiences poverty, then
- Onset of schizophrenia occurs (i.e., a decline in health).

In both models, the distal cause, (e.g., genetic predisposition toward schizophrenia), is a necessary step in the causal stream. However, in neither model is having the genetic predisposition sufficient. In the Social Causation Model, being vulnerable to schizophrenia is insufficient without exposure to poverty; whereas in the Selection Model, being vulnerable to schizophrenia is insufficient without the actual onset of schizophrenia. In other words, only if the distal cause is present can the proximal causes proposed by selection, causation, and confounding theories be relevant within the causal stream. It is in this sense we suggest interpreting the findings presented in this article.

The prior illustration used schizophrenia, but the logic holds for all heritable traits. However, Adler and others have framed genetic effects as caused by third variables. There are many traits with substantial heritabilities that could be "third variables" and more proximate causal influences on health. Accordingly, more research should be dedicated to identifying what these third variables are. A reasonable starting point would be examining whether (and which) individual differences in personality and cognitive ability explain the identified genetic overlap. Recent work identifies that genes are a large source of covariation for health and SES with intelligence (see Arden et al., 2016; Marioni, Davies, et al., 2014; Marioni et al., 2016; Trzaskowski et al., 2014). Future work should test whether those common genetic sources are common to one another, using models and analytic approaches similar to those in the current study.

If measures of individual differences in personality are the third variables at the heart of the gradient, then recent advances in research on personality change and growth-mindset interventions can potentially provide the groundwork for larger scale interventions (Magidson, Roberts, Collado-Rodriguez, & Lejuez, 2014; Paunesku et al., 2015). Just as the impact of phenylketonuria (PKU)—a genetic metabolic disorder—can be circumvented with changes in diet (Jahja et al., 2017; National Institutes of Health, 2000; Plomin, DeFries, & McClearn, 1990), the gradient can potentially be affected by adapting personality change interventions. Consequently, the bivariate heritability gradient should decline with time because postintervention, there would be more homogeneity in people acting in health-preserving and wealth-generating manners (i.e., the phenotypic correlation would reduce; thus, causing heritability to go down with it).

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Caveats and Weaknesses

Measures. Our measures of health used were not diagnosis-specific, but rather were overall measures of health quality and functioning. Accordingly, specific symptoms, clinical diagnoses, and overall mental quality potentially have different distributions of genetic and environmental influences, which may in turn map onto different theoretical pathways. For example, although many measures of mental health symptoms, diagnoses, and overall quality have a shared-environmental component (Burt, 2009; Polderman et al., 2015), including the SF-12's measure of mental health (Steenstrup et al., 2013), their genetic influences vary. For example, clinical diagnoses of major depressive disorder have a well-established genetic component (Polderman et al., 2015; Sullivan, Neale, & Kendler, 2000), especially in clinical populations, whereas measures of depressive symptomology (Byers, Levy, Kasl, Bruce, & Allore, 2009) appear to lack a genetic influence in the general population. Thus, depressive symptomatology may share variance through the shared-environment, whereas major depressive disorder may share variance through genetic sources. Again, further research is needed to untangle the mechanisms for specific disorders.

There is no single definitive measure for SES. Many studies referenced throughout this article have used a single component of SES (e.g., household income, or education) at a given age, and used it as a proxy for the entire construct over all time periods.

Some studies, notably Lichtenstein and colleagues (1993) report results using multiple components of SES, but still at a lone time point and with a focus on each component separately from the others. Combined, these studies cast a nomological net, allowing us to understand the interrelated nature of SES. This article's purpose, however, was to understand the biometrical underpinnings of the SES-health gradient—not the gradient in relation to separate SES-components. Thus, we made a deliberate choice to focus on the higher-level SES construct. Hence, we used an overall index (Myrianthopoulos & French, 1968; Turkheimer et al., 2003) rather than to conduct repeated analyses on separate components of SES. Further research is needed to examine which components of SES contribute to the overall relationship between SES and health.

Power. The nature and number of kinship categories have mixed impact on the power of this study, relative to classic twin designs. Unlike classic twin studies where monozygotic and dizygotic twins are used exclusively, the bulk of subjects in our sample were either full- or half siblings. For a given design and sample size, any study using twins has more power than a study using full and half siblings. The difference in power arises from the larger genetic difference between monozygotic twins ($r = 1$) and dizygotic twins ($r = .5$) relative to the genetic difference between full-siblings ($r = .5$) and half siblings (.25). This relative genetic difference on power is especially impactful for detecting gene-by-environment interactions (Martin, Eaves, & Heath, 1987; Van Der Sluis, Dolan, Neale, & Posthuma, 2008; Van Der Sluis, Posthuma, & Dolan, 2012). Accordingly, we did not even attempt to examine continuous gene-by-environment interactions, as we lack the statistical power to do so. However, unlike classic twin designs, we were not limited to two kinship categories. Across our study, we used additional categories, ranging from monozygotic twins ($r = 1$) to cousins raised in the same home ($r = .125$). These additional categories facilitated joint tests of larger multivariate models. Multivariate models have greater power than the univariate ACE models on which they are based (Schmitz, Cherny, & Fulker, 1998).

We conducted power simulations that suggested that some of our analyses might be somewhat underpowered. This reduced power is irrelevant for the most important findings in our study, the significant overlap in genetic variance for SES and physical health (PCS), and the significant overlap in shared-environmental variance for SES and mental health (MCS). Finding significant results obviates concerns over issues of Type II errors, and reduces concerns with power. Nevertheless, the size of the effect may be smaller upon replication (Button et al., 2013; Ioannidis, 2005). Specifically, although we replicated the shared-environmental correlation between SES and mental health, that effect could still be subject to the "winner's curse" (Button et al., 2013). Indeed, we suspect that the bivariate environmentality for mental health and SES could well be lower in replications because it is a proportion. In contrast, our null findings (no genetic overlap between SES and MCS, and no shared environmental overlap between SES and PCS) could be viewed as suspect because of relatively low statistical power. Nevertheless, a number of supporting design features add to our confidence in these findings, including concurrent validity in relation to other research findings, and replicated results in sensitivity analyses.

We emphasize that the unbalanced sample sizes across kinship categories were the natural consequence of our study's sampling design. The nationally representative nature of this sample has enhanced the study's external validity and has had a mixed effect on power.

Design and statistical modeling. Our findings offer a snapshot of the United States population at age 40, between 1998 and 2006, as portrayed by the NLSY79 data. The effects may vary by age. For example, the shared environment (and the theories explained by the shared environment) may be a more potent influence on the gradient for younger individuals because their childhood experiences are more salient. We plan to replicate and incorporate repeated measures once the Bureau of Labor Statistics (BLS) finishes processing data on health at age 50 in 2016 (and releases the data around 2020). At that point, using more informative designs or complex modeling methods (e.g., Agent Based Modeling; Auchincloss, Riolo, Brown, Cook, & Diez Roux, 2011; Maglio, Sepulveda, & Mabry, 2014), we can more explicitly untangle whether the environmental influences are driven by selection or causation.

Conclusion and Summary

This article decomposed the relationship between SES and health into its biometrical components. The results differed by measure of health. Physical health's relationship with SES was primarily explained through genes, whereas mental health's relationship with SES was primarily explained through the shared environment (and, in each case, by some nonshared environment as well). If we interpret these findings through the genes versus environment framework that theorists, such as Adler and colleagues (Adler et al., 1994; Adler & Stewart, 2010) impose, then these results imply the following:

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- The physical health gradient is a product of social confounding, and is caused by third variables, such as intelligence and personality;
- The mental health gradient is a product of social causation, and SES causes disparities in mental health.

However, we propose that such an interpretation is overly simplistic. Rather, we interpret that the results suggest genetic precursors that are common to both physical health and SES, and shared-environmental influences that are common to both mental health and SES. These genetic precursors do not necessarily imply that third variables are the cause of the SES-physical health gradient. Following the same logic, these shared-environmental influences do not necessarily imply SES causes disparities in mental health. We integrate Adler and colleague's (1994) interpretations with behavior genetics to conclude the following: At age 40,

- The physical health gradient has genetic precursors, that potentially are explained by third variables, such as intelligence and personality;
- The mental health gradient has shared-environmental sources, which is suggestive of a social causation model.

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